show any abnormality. Carcinoma of the breast was diagnosed and a mammogram was performed. This showed a large circular mass with increased density, homogeneous and with well-demarcated borders, compressing the surrounding breast tissue. There was no thickening of the skin. Aspiration produced from a tense cyst about 70 ml of chocolate-coloured fluid containing flakes of tissue. About 50 ml of air was injected and a contrast mammogram was taken with the patient erect.[2] It showed a fluid level and confirmed the diagnosis of a cyst (figure). This was removed intact leaving a rim of atrophied breast tissue. Macroscopically the cyst was very tense, globular, and of about 10 cm in diameter. The outer wall was shaggy. It was multiloculated and contained chocolate-coloured fluid with whitish tissue debris. Histologically it was a typical epidermal cyst lined with stratified squamous epithelium and the cavity contained laminated layers of keratin.

### Comment

Epidermal cysts are usually located on the exposed surfaces of the body. They are filled with keratin,which is often arranged in laminated layers. They commonly result from trauma (including insect bites) or inflammatory down growth with separation and eventual isolation and encystment of a fragment of epidermis. Our case is unusual in three respects. (1) Though small sebaceous cysts are not uncommon in the skin over breast, reports of large epidermal cysts are rare. (2) Multiloculated cysts are uncommon. (3) The cyst's tenseness and firmness gave an appearance of malignancy, which routine mammography seemed to confirm. Luckily, aspiration disclosed the cystic nature of the tumour and air-contrast mammography clinched the diagnosis. The case also illustrates the value of air-contrast mammography in any doubtful cystic lesion of the breast.

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**South Eastern Railway Hospital, Calcutta - 700 043, India**
P K CHATTERJEE, MD, DCP, senior pathologist
S N ROY, MD, DMRD, senior radiologist

# Comparison of effects of propranolol and metoprolol on airways obstruction in chronic bronchitis

Beta-adrenergic blockade is an established treatment for hypertension and angina pectoris; coexisting chronic obstructive bronchitis is common in such patients. Propranolol[1] and metoprolol[2 3] have been assessed in patients with bronchial asthma but there is little information on the effect of metoprolol on the airways of patients with chronic bronchitis.[4] I report a study on the effect of intravenous propranolol and metoprolol on airways obstruction and the response to inhaled isoprenaline in such patients.

### Patients, methods, and results

Ten patients with a mean age of 63 years were studied. All were cigarette smokers with symptoms of longstanding chronic bronchitis, a mean forced expiratory volume in one second ($FEV_1$) of less than 70% predicted value, negative skin tests, and without blood eosinophilia. They attended at the same time of day on three occasions, having had no bronchodilator drugs for 10 hours. After 20 minutes' rest $FEV_1$, forced vital capacity (FVC), specific airways conductance (SGaw), and pulse were measured twice to ensure a stable baseline. Propranolol (0·06 mg/kg, mean 3·8 mg), metoprolol (0·12 mg/kg, mean 7·6 mg), and 0·9% saline were given by slow intravenous injection in a double-blind random order. The measurements were repeated 15, 30, 45, and 60 minutes after the drug. Thereafter 0·16 mg of aerosol isoprenaline sulphate was administered and the measurements repeated 10 minutes later.

The results (see table) were analysed using a paired sample Wilcoxon test. After propranolol there was a significant fall in $FEV_1$ and FVC throughout the hour of study. Metoprolol produced no significant change in $FEV_1$ but a fall in FVC. On comparing the two drugs the fall in $FEV_1$ and FVC at 15 minutes after propranolol was significantly greater than after metoprolol ($P<0·05$). Both drugs produced a significant fall in pulse rate ($P<0·002$). $FEV_1$ improved after aerosol isoprenaline in each group ($P<0·01$). With propranolol, however, it did not reach the pretreatment measurement. The final measurements of $FEV_1$ and FVC with propranolol and isoprenaline were significantly lower than with placebo and isoprenaline ($P<0·01$) or with metoprolol and isoprenaline ($P<0·05$). Mean SGaw improved after isoprenaline in each group ($P<0·01$), but the improvement was significantly greater with metoprolol than it was with propranolol ($P<0·02$).

*Mean $FEV_1$ and FVC (litres), SGaw ($s^{-1}$ $kPa^{-1}$), and pulse (beats/min) before treatment and after aerosol isoprenaline, and the changes after intravenous drugs*

| Treatment | Mean values before intravenous drugs | Mean changes after intravenous drugs at: +15 | +30 | +45 | +60 min | Mean values after aerosol isoprenaline |
|---|---|---|---|---|---|---|
| Placebo | $FEV_1$ 1·34 | −0·04 | 0 | +0·01 | −0·02 | 1·47 |
| | FVC 2·79 | −0·02 | +0·05 | +0·12 | +0·07 | 3·10 |
| | SGaw 0·50 | 0 | +0·02 | +0·03 | +0·03 | 0·70 |
| | pulse 77 | −3 | −4 | −5 | −5 | 73 |
| Propranolol | $FEV_1$ 1·33 | −0·20* | −0·14† | −0·15* | −0·12† | 1·30 |
| | FVC 2·77 | −0·37‡ | −0·28‡ | −0·20‡ | −0·15* | 2·79 |
| | SGaw 0·45 | −0·07 | −0·04 | +0·07 | −0·01 | 0·53 |
| | pulse 80 | −14 | −17 | −16 | −17 | 64 |
| Metoprolol | $FEV_1$ 1·29 | −0·07 | −0·07 | −0·06 | −0·03 | 1·43 |
| | FVC 2·75 | −0·14† | −0·22† | −0·18‡ | −0·12 | 3·00 |
| | SGaw 0·45 | −0·03 | −0·01 | +0·05 | 0 | 0·69 |
| | pulse 79 | −16 | −16 | −18 | −16 | 63 |

On comparison with placebo: *$P<0·02$. †$P<0·05$. ‡$P<0·01$.
*Conversion: SI to traditional units*—SGaw: $s^{-1}$ $kPa^{-1} \approx s^{-1}$ $cmH_2O^{-1} \times 10$.

### Comment

Sympathetic activity is increased in patients with chronic bronchitis; consequently there is a risk of deterioration in symptoms and increased airways obstruction when beta-blockers are used.[5] Two patients had increased dyspnoea after intravenous propranolol. There was no evidence of cardiac failure and no additional treatment was required. Both patients had initially poor ventilatory function and there was no clinical deterioration on the day they received intravenous metoprolol. Propranolol acts on $beta_2$-receptors in bronchial smooth muscle, producing bronchoconstriction, while metoprolol produces no significant change. Thus in patients with chronic bronchitis metoprolol is less likely to produce deterioration in ventilatory function and clinical symptoms. Nevertheless, since beta-blockers are given by mouth over long periods and at various doses further study of metoprolol under such conditions would be appropriate. It is impossible to predict the effect of beta-blockers on the airways obstruction of individual patients, and whenever possible ventilatory function should be monitored during the initial phase of treatment. I took care in this study to exclude patients with bronchial asthma. A previous report,[3] however, showed that the response to metoprolol is unpredictable in such patients and therefore should be used with caution.

I conclude that metoprolol is a safer drug than propranolol when beta-blockade is required in patients with moderately severe chronic obstructive bronchitis. This is of clinical importance in view of the increasing use of these drugs for angina pectoris and hypertension, diseases commonly associated with chronic bronchitis.

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**Pulmonary Function Laboratory, Department of Clinical Measurement, Ninewells Hospital, Dundee DD1 9SY**
D J M SINCLAIR, MB, MRCP, senior registrar in respiratory medicine